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BIOLOGICAL CONSEQUENCES OF BIOACCUMULATION IN AQUATIC ANIMALS: AN ASSESSMENT OF THE CURRENT LITERATURE

by

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This report presents, in narrative and tabular form, an analysis and the results of a literature search conducted to determine the sublethal effects of environmental pollutants on aquatic organisms. It also identifies those biological response parameters which are to be used for scientific interpretive guidance on the consequences of bioaccumulation.

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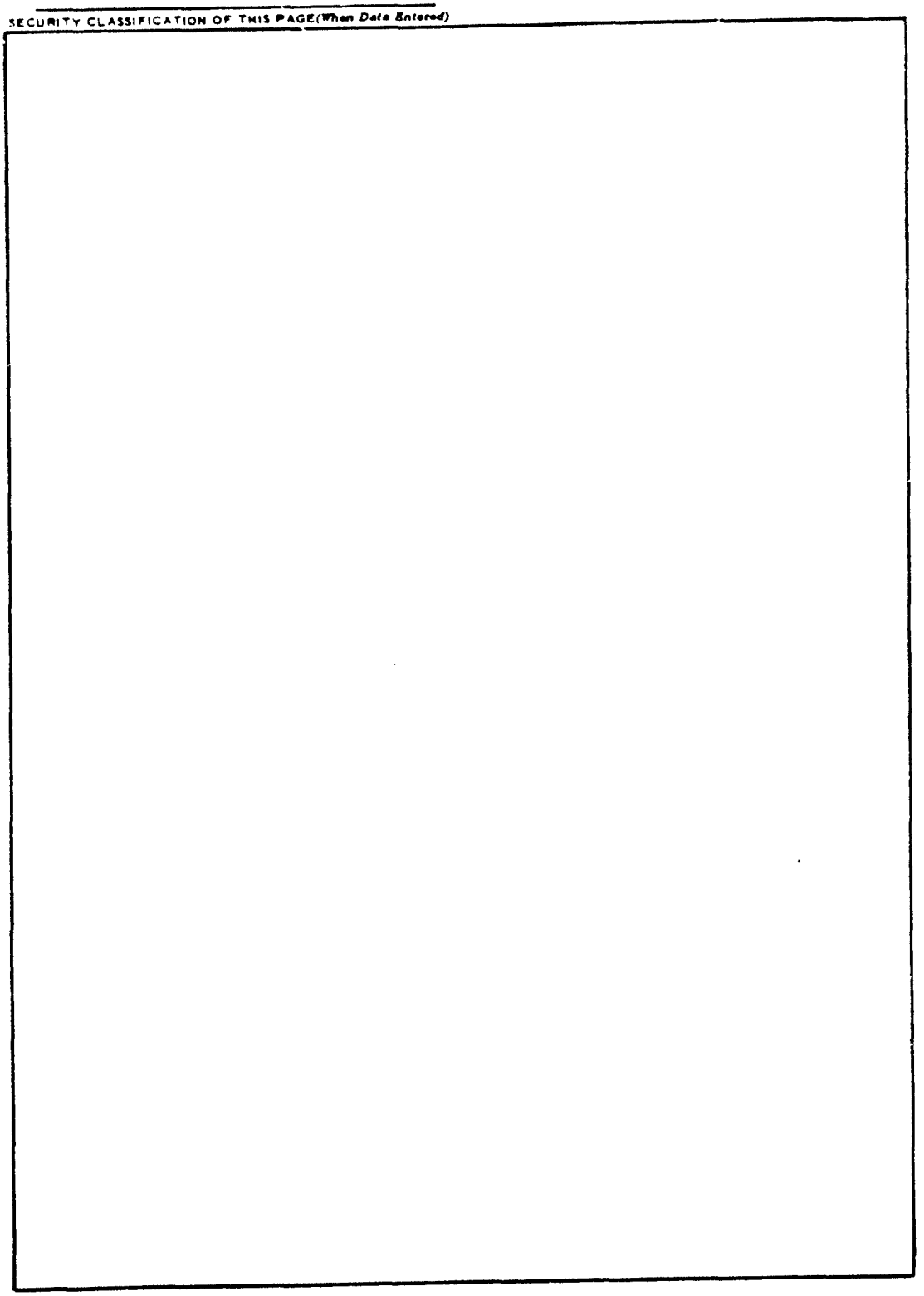

PREFACE

This report discusses results of a review of the open literature conducted at the Environmental Laboratory (EL), U. S. Army Engineer Waterways Experiment Station (WES), Vicksburg, Mississippi, during the period from February 1982 to May 1983. The review was conducted as part of the Long-Term Effects of Dredging Operations (LEDO) Program, which is sponsored by the Office, Chief of Engineers (OCE), U. S. Army. This report represents the initial document produced under Work Unit 31773, Environmental Interpretation of Consequences from Bioaccumulation of the LEDO program.

Section 103 of Public Law 92-532 (Marine Protection, Research, and Sanctuaries Act of 1973) and Section 404 of Public Law 92-500 (Federal Water Pollution Control Act of 1972) require, among other things, that certain ecological evaluations be made prior to disposal of dredged materials in marine or freshwater environments. As part of these evaluations, an estimation of the potential for bioaccumulation of environmental contaminants is often carried out. At present there is insufficient interpretive guidance with which to construe the results of bioaccumulation tests in terms of the potential adverse or unacceptable environmental impact.

Work Unit 31773 was designed, in part, to help provide that interpretive guidance. This report is a review of papers in the open literature dealing with the sublethal biological effects of aquatic organisms contaminated with environmental pollutants. This review was designed to provide an initial source of literature and to help guide future research conducted under this LEDO work unit.

The review was conducted by Dr. T. M. Dillon of the Ecosystems Research and Simulation Division (ERSD), EL. The work was performed under the general supervision of Dr. R. K. Peddicord, Team Leader, Biological Evaluation and Criteria Team, EL, and Dr. R. M. Engler, Group Leader, ERSD, EL. The Chief of ERSD was Mr. D. L. Robey and Chief of EL was Dr. John Harrison. LEDO is managed within EL's Environmental Effects of Dredging Programs, Mr. C. C. Calhoun, Jr., Manager, and Mr. R. L. Lazor, LEDO Coordinator. The Technical Monitors were Dr. John Hall, Dr. William L. Klesch, and Mr. Charles W. Hummer.

Commander and Director of WES during the conduct of this review was COL Tilford C. Creel, CE. Technical Director was Mr. F. R. Brown.

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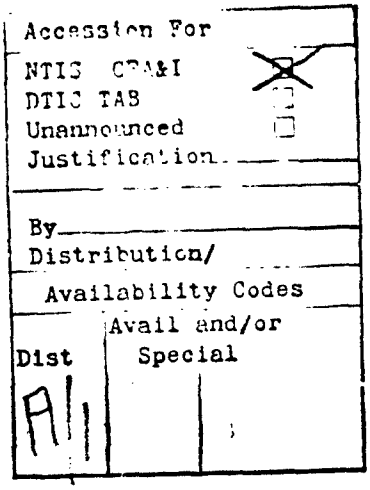

CONTENTS

BIOLOGICAL CONSEQUENCES OF BIOACCUMULATION IN AQUATIC ANIMALS

PART I: INTRODUCTION

Background

1. Approximately 370 million m^3 of sediments are dredged and disposed of every year in the United States (Engler 1980). Approximately half of this volume is disposed of in open water. Because many channels and harbors are located near urban/industrial areas, the sediments to be dredged are contaminated with a variety of environmental pollutants. It is the responsibility of the U. S. Army Corps of Engineers to ensure that these contaminated sediments are dredged and disposed of in a manner which will not result in unreasonable degradation of or an unacceptable adverse impact on the aquatic environment. The two Federal laws by which dredging operations are regulated are Section 404 of the Federal Water Pollution Control Act, as amended (PL 92-500), which deals with inland and estuarine activities, and Section 103 of the Marine Protection, Research, and Sanctuaries Act, as amended (PL 92-532), which regulates ocean dumping.

2. Both of these laws require among other things an estimation of acute sediment toxicity and bioaccumulation potential in aquatic organisms prior to dredging. The former is determined via short-term bioassays while the latter is generally accomplished by chemical analysis of the tissues of organisms surviving the bioassay. The laws and regulations also require that an evaluation be made of the environmental consequences of the observed bioaccumulation, i.e., the biological and ecological effect of specific levels of pollutants in the tissues of organisms exposed to contaminated sediments. This toxicity and bioaccumulation information is used in assessing the potential for toxicological impacts from open-water discharge of the dredged material in question.

3. A variety of investigators have reported that even highly contamined sediments are often not acutely toxic to aquatic animals (DiSalvo et al. 1977, Neff et al. 1978, Shuba et al. 1978). However, accumulation of certain contaminants, such as chlorinated hydrocarbons, is often observed. Therefore,

in the absence of acute toxicity the assessment for potential environmental impact must rely heavily on the bioaccumulation data and its interpretation in terms of potential environmental impact. Unfortunately, there is little generally accepted interpretive guidance regarding the relation between specific levels of tissue contamination and their biological impact (Peddicord and Hansen 1983).

Purpose

4. The purpose of the literature review reported here was threefold. One was to survey the open literature for papers reporting both the sublethal effects of environmental contaminants and the corresponding body burdens in aquatic animals. These papers were reviewed from a broad perspective in order to identify potentially useful information on the relationship between bioaccumulation and biological effect. It is realized that this relationship is an association or, at best, a correlation between two observations. It is not intended nor should it be construed to mean that the relationship between effect and tissue concentration is causal. The second purpose of the review was to identify those biological response parameters which appear to offer the most promise in providing sound scientific interpretive guidance on the consequences of bioaccumulation. The third purpose was to provide an initial source of literature for decisionmakers who have more site-specific concerns (e.g., a particular organism and a specific class of contaminants).

Scope

5. A total of 2181 papers were initially examined in the course of this review. All dealt with the sublethal effects of environmental pollutants on aquatic organisms. However, only 131 papers (6 percent of those examined) reported usable data on the concentrations of contaminants in the tissues of the study organisms. Since a major objective of this study was to examine the relationship between effect and tissue concentration, only these 131 papers were reviewed in detail for this report.

PART II: APPROACH

6. The open literature was searched for papers dealing with the sublethal effects of environmental pollutants on aquatic organisms. Papers dealing with organisms other than macroinvertebrates and fish were not considered because they are not generally used in the regulatory toxicity and bioaccumulation tests. Since one objective of this review was to examine the relationship between bioaccumulation and effect, papers that did not report tissue concentrations in conjunction with biological effects measurements were not reviewed for inclusion in this report. Literature published before the mid- to late 1970s was generally of minimal value since prior to that time emphasis was primarily on the biological effects measurements. Investigators at that time occasionally reported exposure concentrations but only rarely measured tissue concentrations. Since that time, an increasing number of papers have included analytical data in conjunction with effects measurements.

7. For every paper that was reviewed, the following information was recorded: contaminant, test animal, exposure time, exposure concentrations, resultant tissue concentration, and corresponding biological effect. The test animal was identified by a scientific or a common name. Tissue concentrations were all expressed on a wet-weight basis. If the original citation reported tissue concentrations on a dry-weight basis, they were converted to wet weight using the value of 80 percent body water (Emerson 1969, Tucker and Harrison 1974, Florey 1966, Lagler et al. 1962). While the body water percentage of different animals can differ from this representative value of 80 percent, the quantitative variance due to interspecific differences is much less than that due to expressing tissue concentrations on both a wet and dry basis because the majority of animals were exposed to a pollutant in aqueous solution. Exposure concentrations were all given in micrograms per liter (parts per billion) unless noted otherwise.

8. There were nine general categories of sublethal biological responses in the reviewed literature. These were the organismic response parameters of growth, reproduction, morphology/histology, behavior, metabolism, and osmotic/ionic regulation and the biochemical response parameters of enzymes, biochemical composition, and blood chemistry. All citations were initially grouped according to similar sublethal response parameters. Those reports that examine more than one category of sublethal response were included in

each of the appropriate categories. Citations grouped under the same category of biological response were then segregated according to the class of contaminant to which the animal was exposed. Those involving heavy metals appear first, chlorinated hydrocarbons second, and petroleum hydrocarbons last.

9. All citations were examined for the highest tissue concentration at which no biological effect was observed as well as for the lowest tissue concentration at which an effect was observed. These values are referred to as the Highest No Effects Concentration (HNEC) and the Lowest Effects Concentration (LEC). Some papers reported a dose-response and therefore contained both a HNEC and a LEC value. All other papers had only one or the other depending on whether or not a significant response was detected.

10. The HNEC and LEC values were initially used to compare the three classes of contaminants regardless of biological effect. The second comparison, using only the LEC, evaluated the relative sensitivity of the different sublethal biological responses. Papers that reported contaminant concentrations in specific tissues or organs rather than in the whole animal were not included in either of these two comparisons.

11. The HNEC and LEC values were extremely variable, as will be discussed later. For that reason, descriptive statistics (mean, standard deviation, etc.) were calculated, but statistical comparison of the data was not attempted. However, a qualitative trend assessment of the different sublethal responses was made. This assessment was based on the potential usefulness of these biological responses as regulatory tools to aid in the interpretation of the bioaccumulation data required by law.

PART III: RESULTS AND ANALYSIS

Results

12. The citations were initially segregated on the basis of the type of sublethal response examined and are shown in Tables 1-9. Tables 1-6 list those citations that examined the organismic responses of growth, reproduction, morphology/histology, behavior, metabolism, and osmotic/ionic regulation, respectively. Tables 7-9 list those citations that examined the biochemical responses of enzymes, biochemical composition, and blood chemistry, respectively. (Note: Tables 1-9 fulfill the third objective of this study, which was to provide an initial source of literature for those who have site-specific concerns.)

13. The six types of organismic responses represented the majority (75 percent) of table entries while biochemical responses were only 25 percent of the total (Table 10). Growth was the most frequently examined biological response (22 percent) followed by reproduction, morphology/histology, and behavior at 15, 15, and 11 percent of the total, respectively. The use of enzymes as an indicator of biological effect was the most common biochemical response examined (10 percent). The remaining organismic (metabolism and osmotic/ionic regulation) and biochemical (biochemical composition and blood chemistry) responses each represented less than 9 percent of the total table entries.

14. There were three major classes of contaminants in the reviewed literature: heavy metals, chlorinated hydrocarbons, and petroleum hydrocarbons. The frequency of the types of contaminants mentioned in the literature is shown in the following tabulation. Heavy metals were studied in a majority of the papers (53 percent) followed by chlorinated and petroleum hydrocarbons, at 30 percent and 15 percent of the total, respectively. Cadmium was the most

	Contaminant						
Frequency	Heavy Metals	Cadmium	Mercury	Chlorinated Hydrocarbons	PCBs	Petroleum Hydrocarbons	Other
Number of entries	65	25	24	37	20	19	3
Percent of total	53	20	19	30	16	15	2

commonly examined heavy metal (20 percent) while polychlorinated biphenyls (PCBs) were the most frequently studied chlorinated hydrocarbon (16 percent). No one particular type of petroleum hydrocarbon dominated this class of contaminant. Three papers dealt with pollutants that did not belong to any of these three major classes of contaminants (Goodman et al. 1979, Spehar et al. 1983, Wells and Cowan 1982).

15. The HNEC and LEC for all the entries were first grouped according to the type of contaminant regardless of the sublethal response. Both HNEC and LEC are extremely variable regardless of pollutant (Table 11). This variability is seen both in the range of tissue concentrations and in the coefficients of variation, which range from about 85 to 260 percent. There was a slight reduction in this variability when the most commonly examined contaminants (i.e., cadmium, mercury, and PCB) were considered individually. Further attempts to reduce this high variability by considering more specific groupings (e.g., cadmium and arthropods) were impractical due to the limited number of papers. This degree of variability in tissue concentrations makes statistical comparisons of little use and therefore limits the interpretation of the results.

16. The mean HNEC and mean LEC for animals exposed to petroleum hydrocarbons were greater (117-127 µg/g) than for those exposed to metals (31.0-43.5 µg/g), which, in turn, were slightly higher than for those exposed to chlorinated hydrocarbons (14.4-16.4 µg/g) (Table 11). This implies that when a range of sublethal parameters is considered, chlorinated hydrocarbons are somewhat more toxic than heavy metals which are more toxic than petroleum hydrocarbons.

17. The relative sensitivity of the various sublethal responses to all contaminants was evaluated by comparing the mean LEC within each category of response (Table 12). Tissue concentrations again showed high variability, as indicated by the very large range of values for each response and the coefficients of variation, which ranged from about 80 to 280 percent. Papers that evaluated morphological and histological characteristics of contaminated organisms had a very low mean LEC value (7.0 µg/g) relative to the mean value for the other organismic response parameters, which ranged from 26.0 to 76.5 µg/g. This indicates that morphological and histological observations may be sensitive sublethal evaluative tools. It is difficult to interpret the importance of the relatively low mean LEC for osmotic/ionic regulation

(26.0 µg/g) since the sample size was so small (n = 3). The mean LEC value for all biochemical response parameters (31.3 µg/g) was only slightly lower than the overall mean for organismic responses (50.9 µg/g).

18. The frequency of the mode of exposure reported in the reviewed papers is shown in the following tabulation. According to the majority of

	Mode of Exposure				
Frequency	Water	Food	Field Collected	Sediment	Injection
Number of entries	91	19	12	9	4
Percent of total	67	14	9	7	3

papers reviewed for this report (67 percent), aquatic animals were exposed to aqueous solutions of pollutants. Contaminated food was the second most frequent mode of exposure (14 percent). In 9 percent of the papers, animals were collected from naturally contaminated field environments, but the primary route of contamination was not identified. Exposure to contaminated sediments occurred in only 7 percent of the papers reviewed, while hypodermic injection as the mode of contamination occurred in 3 percent (four papers) of the total.

Analysis

Relationship between bioaccumulation/biological effects

19. One objective of this study was to review the current literature for papers describing the relationship between bioaccumulation and biological effect, i.e., the point at which specific tissue concentrations in aquatic animals begin to have an ecologically meaningful effect. There were relatively too few reports in the open literature that contained the necessary data to examine the association of tissue contamination and biological effect. In those reports that did contain both effects and bioaccumulation data, the variability in tissue concentrations was so great that specific conclusions and recommendations could not be made. Moreover, there were too few papers for adequate evaluation of the relationship between specific contaminants and individual groups of animals.

20. There was an indication that the biological changes associated with petroleum hydrocarbons in tissues occurred at higher tissue concentrations

than changes associated with heavy metals or chlorinated hydrocarbons in the tissues. Also, aquatic animals appear to be slightly more sensitive to chlorinated hydrocarbons than to metals. Similar results were obtained in water quality bioassays in which chlorinated hydrocarbons were toxic in the microgram-per-liter range, heavy metals in the milligram- and microgram-per-liter range, and petroleum hydrocarbons in the milligram-per-liter range (U. S. Environmental Protection Agency 1976).

Indicators of biological consequences of bioaccumulation

21. The second major objective of this review was to identify those types of sublethal response parameters that appeared to be promising indicators of the biological consequences of bioaccumulation. Tissue concentrations at which an effect was first observed were too variable to make any quantitative assessment among the different categories of sublethal response.

22. Some qualitative recommendations can be made based on their intrinsic scientific value as well as their potential use by the regulatory community. The latter criterion generally refers to the practicality of the methodology and its ecological interpretability. In this context, potentially useful biological responses are those that can be determined in a reasonable period of time. This criterion is important since the Corps of Engineers processes many permits every year and conducts many Federal dredging operations which, while not requiring a permit, must still comply with the regulations of the Federal Water Pollution Control Act and the Marine Protection, Research, and Sanctuaries Act. Interpretation of the biological response should be as unambiguous as possible and not require intensive specialized training or expertise.

23. There is another criterion for potentially useful responses that is unique to the evaluation of mixtures of pollutants. Since dredged materials often contain a range of different contaminants in various proportions, any sublethal parameter used to evaluate biological effect should be sensitive not only to one class of contaminants but also to the general health and well being of aquatic organisms that have accumulated a wide variety of pollutants.

24. Growth. In all the papers reviewed, growth was the most frequently examined biological response (22 percent). This is probably due, in part, to the fact that it is a relatively easy measurement to make. Growth is usually determined by measuring either a change in mass or a change in some physical

dimension, such as length, after a period of exposure. The measure of growth integrates many facets of the organism's response to pollutant exposure and is therefore a good index of the organism's general health and well being. It generally cannot specify mechanistic causes, but from a regulatory standpoint this may not be necessary. Laboratory determinations of growth can be achieved in a matter of days to weeks depending on the animal and the exposure conditions.

25. The use of growth as an evaulative technique has one major drawback that is common to almost all laboratory-derived data--discriminating between statistically significant differences and observations that are ecologically meaningful. A well-designed and properly performed experiment can often detect small yet statistically significant differences between treatments. It is much more difficult to decide what constitutes a truly ecologically meaningful difference. This is hampered by our lack of knowledge of how altered growth rates determined in the laboratory relate to population dynamics in the field. It is unlikely that this knowledge will be available in the immediate future, and until it is, interpretation of the results of growth studies will have to continue to be based on the best available data and accumulated scientific judgment.

26. Reproduction. Reproduction was the second most frequently examined sublethal parameter in this review (15 percent). Ecologically, it is a very important index because if a species does not successfully reproduce, it will eventually become extinct. What constitutes an ecologically important impairment in reproductive capability is again often a difficult judgment to make. Nevertheless, this response has less ambiguity associated with its interpretation than most other sublethal responses. Reproductive studies are also valuable because they often deal with early life stages of aquatic organisms which are generally believed to be more sensitive to a variety of pollutants than at the more mature stages. Advances in culture techniques have enabled multiple life cycles to be completed in the laboratory in a matter of weeks to months, depending on the organism. Several investigators have concluded that reproductive studies probably yield more valuable insight into the effects of environmental pollutants than any other one approach available today (Klapow and Lewis 1979, Sprague 1971, Cairns et al. 1978, Eagle 1981).

27. Morphology/histology. The morphology/histology parameter was also frequently examined (15 percent) in the papers reviewed. In addition, there

was some indication that it is more sensitive than other organismic response parameters (Table 12). However, this category of response was found to suffer from one primary disadvantage--a lack of quantification. Most of the papers in the open literature, including those reviewed here, report observations such as hemorrhaging, spinal deformities, emaciated and necrotic tissues, hyperplasia, loss of distinctive histological features, fibrosis, etc. These observations were not normally quantified, and they were often made secondary to some other response parameter that the investigator was initially interested in. These secondary morphological and histological observations are often helpful in explaining results obtained with other parameters but have, by themselves, little interpretive value when examining the relationship between effect and bioaccumulation.

28. These observations suffer another disadvantage--a lack of definition for normal histology and cell morphology. This is due, in part, to a lack of precise quantification but is more directly related to our general lack of baseline information in this field of aquatic biology. Finally, an experienced person is required to properly prepare a usable histological sample and to interpret what is being observed. For these reasons, it appears that for the immediate future morphological and histological observations do not hold high potential as a regulatory method for evaluating dredged material.

29. Behavior. This parameter also has many of the disadvantages associated with morphological and histological observations just discussed. Lack of a quantifiable response is the major drawback. Aberrant behavior reported in the papers reviewed was often reported secondary to other parameters under consideration. Behavioral observations can generally be divided into avoidance responses and altered normal behavior following pollutant exposure. These changes, while not usually immediately lethal, may eventually reduce the organism's survival potential. Observations of behavior, in response to pollutant contamination, will increase in value if more studies can be designed specifically to evaluate behavior in response to pollutant contamination. A useful conceptual framework for the experimental approach of such studies has been outlined by Olla et al. (1980).

30. Metabolism. Almost all the papers reviewed that examined the metabolism of exposed aquatic animals (9 percent) measured the consumption of oxygen over time. In those papers, there was no consistent trend of increasing or decreasing oxygen consumption associated with tissue contamination.

This was not too surprising since this parameter is normally quite variable and is affected by a variety of endogenous and external factors as well as pollutants (Newell 1970, Prosser 1973). This variability in oxygen consumption has been cited as the major obstacle in its use as a regulatory tool (Bayne et al. 1980, Dillon and Lynch 1981).

31. Oxygen consumption can be of greater value when measured in conjunction with other elements of metabolism. The best example of its use in this manner is in scope for growth measurements (Bayne 1975, Bayne and Widdows 1978, Widdows 1978). A determination of scope for growth involves measuring various components of metabolism such as feeding rate, ingestion efficiency, and excretion rate, as well as oxygen consumption. The components are all expressed on a caloric basis, which allows an instantaneous energy budget to be calculated. This calculation reveals whether the animal has a surplus of energy (positive scope for growth) or a deficit (negative scope for growth). If the latter condition persists, the animal may die or undergo a reduction in reproductive effort. Scope for growth measurements have been correlated with more traditional physiological indices such as growth and reproduction (Bayne et al. 1978, Bayne and Worral 1980). The laboratory measurements needed to calculate scope for growth require only 24 to 48 hr. For these reasons, scope for growth appears to hold promise for regulatory applications. However, there is one distinct limitation that prevents its widespread immediate use. Scope for growth determinations have been performed almost exclusively on marine mussels. Before it can be more widely applied, the methodology must be developed with a broader range of aquatic animals.

32. Osmotic/ionic regulation. Osmotic and ionic regulation in aquatic animals involves the maintenance of proper water balance and solute concentrations in the body tissues. There are great interspecific differences in the ability of fresh and salt water animals to osmoregulate (Prosser 1973). Consequently, before the association of this sublethal parameter with tissue contamination can be evaluated, the test species' osmoregulatory capability must first be determined. In the regulatory evaluation of dredged material, this collection of baseline information is often logistically and financially impractical. Even if the information on normal solute concentrations and their flux rates can be obtained from the literature, it is unclear what magnitude of deviations from steady-state levels (other than those causing death) are truly harmful to the organism. Finally, measuring osmoregulatory parameters,

such as total osmotic pressure and the flux rate of osmotically active organic and inorganic solutes, is beyond the normal capabilities of many laboratories. All of these disadvantages make this category of response a less attractive regulatory tool and may explain why it was utilized so infrequently (3 percent) in the papers reviewed in this report. These disadvantages have also been reported in the evaluation of different types of physiological responses for use in pollution monitoring programs (Bayne et al. 1980).

33. There is some evidence in the literature which indicates that one specific aspect of ionic regulation may be affected when aquatic organisms are exposed to substances containing chlorine. Roesijadi et al. (1979) and Laird and Roberts (1980) reported a disruption in the regulation of magnesium in marine crabs exposed to chlorinated seawater. The exposed crabs had abnormally high blood levels of magnesium that were positively related to exposure concentration. Caldwell (1974) also found elevated serum magnesium levels in marine crabs exposed to methoxychlor. Magnesium has a direct effect on nerve impulse transmission rates, and elevated levels can result in sluggish behavior and paralysis. These behavioral changes were observed in two of the above reports (Caldwell 1974, Roesijadi et al. 1979). Coupled observations of behavior and magnesium regulation may be useful in situations where a chlorinated pollutant is of major concern.

34. Enzymes. Enzymes constitute a class of biological molecules (proteins) that are essential to all organisms. They act as catalysts, reducing thermodynamic thresholds so that biochemical reactions can occur at a rate that permits life to exist. In spite of the tremendous diversity of living organisms, enzymes and the biochemical pathways they catalyze are remarkably similar. This interspecific similarity and the fact that enzymes are essential to all life theoretically makes them very appealing indicators of pollutant contamination.

35. However, enzyme systems are extremely dynamic in aquatic organisms with both rate functions and concentrations of enzymes varying in response to the needs of the animal as well as to changes in the external environment. There are feedback control mechanisms that function to stabilize individual pathways and the interaction among pathways, thus preventing the excessive accumulation or depletion of organic substrates. Consequently, when a change in a single enzyme is observed in response to pollutant contamination, it is

often difficult to assess what effect this change will have on the overall biochemistry of the organism.

36. There is one specific group of enzymes that shows some promise as an effective monitoring tool. These enzymes are found in lysosomes, the intracellular organelles found in all eucaryotic animals. They contain a wide variety of hydrolytic enzymes capable of breaking down most known biomolecules (Dingle 1973). One of their functions is the controlled degradation of cellular components and tissues, which occurs continuously in all animals.

37. Exposure to environmental contaminants can alter the permeability of lysosomal membranes resulting in the uncontrolled release of their hydrolytic enzymes. If contaminant exposure is severe enough or of sufficient duration, extensive tissue necrosis and eventually death of the organism may result. A reduction in lysosomal membrane stability followed by the release of hydrolytic enzymes has been documented in aquatic animals exposed to heavy metals as well as chlorinated and petroleum hydrocarbons (Harrison and Berger 1982, Moore 1979, Rogers et al. 1976, Viarengo et al. 1981b). In one instance, a decrease in permeability and subsequent release of enzymes has been correlated with a decrease in the general health of oil-exposed marine mussels (Widdows et al. 1982, Table 7).

38. The fact that lysosome membrane stability appears sensitive to a wide variety of contaminants makes it an attractive monitoring tool. However, the present methodology requires a fairly sophisticated histochemical technique. Also, since most work to date has been performed on marine mussels, the dynamics of the lysosomal system need to be examined in a greater number of aquatic organisms before it can be used in an applied regulatory situation.

39. Biochemical composition. Citations that were placed in the biochemical composition category reflected a wide range of types of measurements. Consequently, generalities about their overall usefulness were difficult to make. They did share one common disadvantage that is inherent in many biochemical measurements--the difficulty of relating changes in one specific biochemical parameter to a change in the general health of the individual. There was, however, one type of response listed in this category which merits further discussion.

40. Adenylate energy charge (AEC) is the molar ratio of intracellular adenylates and is calculated by the following equation (Atkinson 1971):

$$AEC = \frac{ATP + 1/2\ ADP}{ATP + ADP + AMP}$$

Adenylates, primarily ATP, are used as an energy source in the cells of organisms. Enzyme-catalyzed biochemical reactions may be generally classified as those requiring energy (ATP) and those producing energy. Because both types of reactions are occurring at the same time, the cell must constantly balance ATP utilization with its production. The AEC is a measure of this balance.

41. It has been argued that AEC is an effective index with which to assess the sublethal effects of pollutants (Ivanovici 1980, Ivanovici and Wiebe 1981). It measures the general energy balance of living organisms and therefore reflects the animal's integrated response to environmental perturbations. It has the potential for wide application and interspecific comparisons because it measures something common to all animals (relative adenylate concentrations).

42. AEC values greater than 0.8 are believed to represent good health, while values less than about 0.7 are considered indicative of deteriorating health. This quantitative response in relation to biological health is very attractive from a regulatory point of view. However, these values have been established primarily from studies with microorganisms and mammalian tissue culture (Atkinson 1971). AEC values for aquatic organisms appear to be much less conservative in that they vary appreciably from tissue to tissue and often do not correlate with other, more traditional physiological measurements (Bakke and Skjoldal 1979, Ellington 1981, Giesy and Dickson 1981, Romano and Daumas 1981).

43. The experimental expertise required to obtain accurate AEC values is substantial because minor changes in the way an organism is maintained or handled can dramatically affect the resultant adenylate concentrations. In addition, there is still considerable debate as to how different tissues should be extracted, stored, and analyzed (Karl and Holm-Hansen 1978, Mendelssohn and McKee 1981, Wadley et al. 1980). One last disadvantage to this parameter is that relatively few studies have examined AEC in aquatic animals exposed to environmental pollutants.

44. Blood chemistry. In both mammalian physiology and human medicine, chemical characteristics of the blood provide an extremely accurate insight into the physiological health of the whole organism. These parameters are

useful because in the above fields of study they have a known predictive value with relatively small deviations from normal values reflecting a potential pathological condition. The predictive value of these blood parameters reflects the fact that mammals tend to maintain a homeostatic or constant internal environment. Fish and aquatic invertebrates, on the other hand, do not normally maintain a strict internal steady state. Rather, when changes in the external environment occur, their physiological systems respond within the organism's individual capability but do not necessarily maintain homeostasis (Mangum and Towle 1977). Consequently, relatively large variations in blood parameters of aquatic animals are not uncommon and reflect normal changes in the organism's internal physiology. This variability greatly reduces the value of blood chemistry parameters and makes associations of changes with tissue contaminant levels difficult to detect.

45. Biological organization. It may be useful to discuss the different levels of biological organization as they relate to the preceding discussions on different sublethal response parameters. There are four general levels of biological organization--biochemical, organismic, population, and community (Capuzzo 1981). It is a generally held belief that when an effect is observed at one level of biological organization, the mechanistic explanation is found at the level below and the environmental implication at the level above (Sprague 1971). From an ecological perspective, only effects at the population and community level are of any importance. However, current regulatory testing is generally conducted at the organismic level.

46. Studies that examine populations and communities vary greatly, but all examine one or two things--structure and/or function. The former refers to species richness and diversity while the latter refers to parameters such as energy flow, nutrient cycling, predation, competition, and other biological interactions. These types of studies have provided good insight into how populations and communities react both to natural and man-induced perturbations. They have also demonstrated that populations and communities are extremely complex and dynamic systems. These characteristics have generally made it very difficult to differentiate the effects of pollution-related perturbations from natural variations. It is usually not until populations and communities are in the latter stages of degradation that pollution-related effects can be clearly demonstrated. A more sensitive means of early detection prior to wholesale degradation is therefore highly desirable.

47. Biochemical parameters are theoretically the most sensitive level of biological organization at which pollutant-induced changes may be detected. It is believed that most environmental perturbations initially impact biochemical processes (Rosenthal and Alderdice 1976). It is this sensitivity plus the fact that biochemical determinations often require substantially less time to perform than population/community studies that make biochemical indices very attractive evaluative tools. However, biochemical studies all suffer one major disadvantage: it is exceedingly difficult to relate a change in some biochemical parameter to the whole organism, much less to a population or community. I am unaware of any study that has demonstrated this causal relation in a quantitative fashion.

48. Investigations at the organismic level of organization appear to be a reasonable compromise between the advantages and disadvantages of biochemical and population/community studies. MacMahon et al. (1978) and MacIntyre et al. (1978) have convincingly argued the advantages of evaluating the effects of environmental perturbations at the organismic level of biological organization. This approach offers greater sensitivity and generally requires less effort than population/community studies. It is also more readily interpreted in terms of potential ecological effect than biochemical studies. In addition, organismic investigations can probably be performed by a greater number of facilities than either biochemical or population/community studies. These advantages, however, are contingent on the specific types of sublethal response that is selected for study. For the reasons given earlier, reproduction, growth, and some measure of general metabolism, such as scope for growth, appear to be the more promising organismic response parameters with which to evaluate the relationship between biological effects and tissue contamination. Similar recommendations have been made in reviews of sublethal effects of pollutants on aquatic organisms (Anderson 1977, Bayne et al. 1980, Dillon and Lynch 1981, MacIntyre et al. 1978, Sprague 1971).

49. Metallothioneins. One objective of this review was to evaluate the relationship between tissue concentrations and biological effect in aquatic organisms. There is a specific biochemical process that may substantially influence this relationship. Vertebrates are known to synthesize intracellularly low molecular weight proteins called metallothioneins in response to heavy metal contamination (Kojima and Kagi 1978). Because of the high sulphur content of these proteins, they readily bind heavy metals, thereby reducing

their potential toxic effects. If the production and chelating properties of these proteins are exceeded by the total metal body burden, the excess metals spillover into the cytosol and exert their toxic effects on intracellular enzymes and membranes.

50. Metallothionein-like proteins have recently been discovered in aquatic invertebrates (see Roesijadi 1981 for review). They appear to function in the same way as vertebrate metallothionein-like proteins and are now believed to explain the observation that aquatic animals often exhibit enhanced heavy metal tolerance following exposure to sublethal concentrations (Pruell and Engelhardt 1980, Roesijadi et al. 1982). The spillover phenomenon, first documented in vertebrates, has been examined in only a few aquatic organisms (Brown and Parson 1978; Roesijadi 1981). The production of metallothionein-like proteins has been correlated with the initiation of tissue repair and regeneration in a marine organism contaminated with copper (Young and Roesijadi 1983).

51. The vast majority of papers in the literature do not discriminate between free metals and those bound to protective metallothionein-like proteins. This may be quantitatively important since metals bound to metallothionein-like proteins can represent 40 to 60 percent of the total body burden (Brown and Parsons 1978, Olson et al. 1978, Pruell and Engelhardt 1980; Roesijadi et al. 1982). If metallothionein-like proteins and the spillover phenomenon are common occurrences in aquatic invertebrates, the analysis of subcellular distributions of metals may become a necessity if associations between body burden and biological effect are to be made accurately.

PART IV: SUMMARY AND CONCLUSIONS

52. The first objective of this review was to gain an insight into the relationship between bioaccumulation and subsequent biological effect in aquatic organisms. In the literature initially examined for this report, only about 6 percent of the papers contained both biological effects and bioaccumulation information. In those papers, the tissue concentrations reported were too variable to make any recommendations regarding the biological consequences of specific tissue concentrations.

53. The second objective of this review was to identify sensitive and potentially useful parameters with which to evaluate the consequences of bioaccumulation. None of the organismic or biochemical response parameters, except morphology/histology, appeared more sensitive than did any other based on the reported lowest tissue concentrations at which an effect was first observed. The response parameters of growth, reproduction, and some measure of metabolism, such as scope for growth, did appear to hold the most potential for immediate use in a regulatory situation.

54. Tables 1-9 contain literature sources that address site-specific concerns such as a particular organism and a specific class of contaminant, which was the third objective of this review.

55. Based on the reported lowest tissue concentration at which an effect was observed, aquatic animals appear to be more sensitive to chlorinated hydrocarbons than to petroleum hydrocarbons with the effects of heavy metals being intermediate.

56. A majority (67 percent) of the papers reviewed for this report involved the study of pollutants in aqueous solution. Only a small minority of papers (7 percent) evaluated biological effects after exposure to contaminated sediments. This disproportion of papers suggests that more effort should be directed at evaluating the biological effects of contaminated sediments if the regulatory responsibilities of the Corps of Engineers are to be addressed.

57. Studies conducted at the biochemical level of organization offer the potential advantage of greater sensitivity but cannot generally be related to effects on populations and communities. From an ecological perspective, the effects on populations and communities are of concern. However, field studies at this level of biological organization are rarely sensitive enough

to detect the early or subtle effects of pollution. Evaluation of the effects of environmental contaminants at the organismic level of biological organization is a reasonable compromise in sensitivity and ecological interpretation.

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Table 1

References in Which Growth Was Evaluated in Aquatic Animals Contaminated with Environmental Pollutants

Reference	Contaminant	Organism*	Exposure Time	Exposure Concentration**	Tissue Concentration†	Biological Effect
Hatakeyama and Yasuno 1982	Cadmium	Guppy (Fw)	25 days	5-10	2.6-5††	No effect on growth
			30 days	69-171 µg/g dry food	0.6-1.2††	No effect on growth
Benoit et al. 1976	Cadmium	Trout (Fw)	156 weeks, 3 generations	0-1.7	No data	No effect on growth
				3.4	1-10 (range in gill, kidney, liver)	Growth reduced in 2nd and 3rd generations
				6.3	No data	100% mortality
Rombough and Garside 1982	Cadmium	Salmon (Fw)	451 days	0.47-300	0.005-2.00††	Growth reduced at all concentrations
Spehar 1976	Cadmium	Flagfish (Fw)	100 days	0-8.1	0-10	No effect on growth
				16-31	20-35	Reduction in growth
Hatakeyama and Yasuno 1981	Cadmium	Arthropod (Fw) (Cladocera)	14 days	14.3-71.2 µg/g dry food	200-225††	No effect on growth or survival
				341.3 µg/g dry food	450††	Decreased growth and survial
Marshall 1978	Cadmium	Crustacean (Fw)	22 weeks, 7 generations	1-4	3.5-8.6††	No effect on growth
				8	10.3††	Enhanced growth
Thorp et al. 1979	Cadmium	Crayfish (Fw)	5 months	0-10	0-22	No effect on growth
Wald 1982	Cadmium	Oyster (M)	8 weeks	25	46††	No effect on growth
			16 weeks	10	25††	Reduction in growth
Poulsen et al. 1982	Cadmium	Mussel (M)	17 days	0-109	0-30††	No effect on growth
Snarski and Olson 1982	Mercury	Minnows (Fw)	31 weeks	0-0.50	0-2.84	No effect on growth
				1.02-3.69	4.47-18.8	Reduction in growth
McKim et al. 1976	Mercury	Trout (Fw)	144 weeks	0-0.29	0-3.4	No effect on growth
				0.93	9.4	Reduction in growth
				2.93	No data	Very high mortalities

(Continued)

* Parenthetical entries after name of organism are defined as follows: (Fw) = Freshwater; (M) = Marine.
** Entries for exposure concentration are in units of micrograms per liter (µg/ℓ) unless noted otherwise.
† Entries for tissue concentration are in units of micrograms per gram (µg/g) wet weight whole animal unless noted otherwise.
†† Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 1 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Koeller and Wallace 1977	Mercury	Salmon (M)	62 days	1-5	0.002-0.009 (liver)	Reduction in growth
Brown and Parsons 1978	Mercury	Zooplankton (M)	62 days	1-5	0.02-0.04 (whole tissues	Reduction in growth
Holcombe et al. 1979	Zinc	Trout (Fw)	136 weeks	0-534	0-30††	No effect on growth
				1360	No data	Very high mortalities
Pierson 1981	Zinc	Guppy (Fw)	134 days	0-607	0-0.3††	No effect on growth
Spehar 1976	Zinc	Flagfish (Fw)	100 days	0-26	0-150	No effect on growth
				51	200	Growth reduced in females only
Farmer et al. 1979	Zinc	Salmon (Fw)	61 days	0-740	0-42	No effect on growth
Holcombe et al. 1976	Lead	Trout (Fw)	144 weeks	0-119	0-40†† (range in liver, gill, and kidney)	No effect on growth
				235	14-60††	Reduction in growth
				473	No data	Very high mortalities
Benoit 1975	Copper	Bluegill fish (Fw)	88 weeks	0-77	0-11.4†† (range in liver, gill, and kidney)	No effect on growth
				162	2.6-96††	Reduction in growth
Stebbing and Pomroy 1978	Copper	Coelenterate (Fw)	11 days	0-2.5	0-3.6††	No effect on growth
				5-10	4.2-9	Reduction in growth
				25-50	No data	High mortalities
Dixon and Sprague 1981	Arsenic	Trout (Fw)	21 days	2950	3.0	No effect on growth
Hansen et al. 1976	PCB (Aroclor 1242)	Catfish (Fw)	20 weeks	20 µg/g dry food	14	Reduction in growth
Bengtsson 1980	PCB (Clophen A50)	Minnow (Fw)	40 days	0-270 µg/g dry food	0-21	No effect on growth
				2500 µg/g dry food	180	Enhanced growth
Mac and Seelye 1981	PCB (Aroclor 1254)	Trout (Fw)	52 days	0.05 µg/ℓ plus 1.0 µg/g dry food	1.8-2.4††	Enhanced growth

(Continued)

†† Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 1 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Anonymous 1981	PCB (Aroclor 1254) + DDE	Trout (Fw)	6 months	10 ng PCB/ℓ + 1.0 ng DDE/ℓ + 1.0 µg PCB/g food + 0.1 µg DDE/g food	1.53-26.3 PCB 0.29-2.68 DDE	No effect on growth at any exposure concentration
				5X the above dose		
				25X the above dose		
Westin et al. 1983	PCB	Striped bass (Fw)	20 days	0.014-0.127 µg/g wet food	0.1-1.8	No effect on growth
Buckler et al. 1981	Kepone	Minnow (Fw)	120 days	0-0.31	0-0.21	No effect on growth
Hansen et al. 1977a	Kepone	Sheepshead minnow (M)	36 days	0.08-6.60	1-22	Growth inversely related to concentration
Goodman et al. 1982	Kepone	Sheepshead minnow (M)	160 days	0-0.12	0-0.86	No effect on growth
				0.39-0.78	1.1-5.0	Reduction in growth
Schimmel et al. 1979	Kepone	Blue crab (M)	28 days	0.15 µg/g food	0.069	Reduction in growth
Fisher 1980	Kepone	Blue crab (M)	65 days	0.36-2.5 µg/g food	0.38-4.61	No effect on growth; ratio of carapace thickness to width inversely proportional to concentration
Buckler et al. 1981	Mirex	Minnow (Fw)	120 days	0-34	8-151	Enhanced growth
Bookhout et al. 1976	Methoxychlor	Crab (M)	10 days	0.7	0.51	Reduction in growth
Oladimeji and Leduc 1975	Methoxychlor	Trout (Fw)	30 days	0.67 µg/g fish/day	0.3-2.5	Slight reduction in growth
Spehar et al. 1983	Permethrin (Synthetic pyrethroid)	Minnow (Fw)	32 days	0.11-0.66	0.19-2.16	No effect on growth
				1.40	4.51	Reduction in growth
	AC 222, 705 (Synthetic pyrethroid)	Minnow (Fw)	32 days	0.02-0.07	0.06-0.17	No effect on growth
				0.13-0.29	No data	Reduction in growth; high mortalities

(Continued)

Table 1 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Hansen et al. 1977b	Endrin	Sheepshead minnow (M)	22 weeks	0-0.31	0.94	No effect on growth
Gunkel 1981	Atrazine	Whitefish (M)	25-30 days	300	0.48	Reduction in growth
				600 µg/kg food	0.25	Reduction in growth
Mayer et al. 1978	Toxaphene	Catfish (Fw)	150 days	0.037-0.068	0.8-1.2	No effect on growth
				0.106-0.475	1.8-14	Reduction in growth
Poels et al. 1980	Rhine River water	Trout (Fw)	18 months	No data	1.6-PCB 0.12-DDE 0.11-Dieldrin 2.5-PCP	Reduction in growth
Hannah et al. 1982	Benzo(a) pyrene	Rainbow trout (Fw)	36 days	2.4	12.34	Reduction in growth
Woodward et al. 1981	Wyoming crude oil	Trout (Fw)	90 days	100-520	14-30 (Accumulation of mono and diaromatics)	Growth inversely proportional to oil concentration
McCain et al. 1978	Alaskan North Slope crude oil	Flatfish (M)	4 months	400-700 µg/g dry sediment	0.037ff after 51 days (liver) (Accumulation of alkylated mono and diaromatics)	Reduction in growth
Clement et al. 1980	Prudhoe Bay crude oil	Bivalve (M)	180 days	0-300	0-700	No effect on growth; condition index decreased
Stekoll et al. 1980	Prudhoe Bay crude oil	Bivalve (M)	180 days	3000	1000 (Accumulation of larger and more highly substituted aromatics)	Reduction in growth; condition index decreased
Augenfield et al. 1980	Prudhoe Bay crude oil	Bivalve (M)	54 days field exposure	850-1237 µg/g sediment	1.77 di and triaromatics 0.337 saturates	Condition index reduced
Roesijadi and Anderson 1979	Prudhoe Bay crude oil	Bivalve (M)	55 days lab exposure	616-1233 µg/g sediment	5.21 diaromatics 0.14 aliphatics	Condition index unaffected
			38 days field exposure	364-1144 µg/g sediment	0.46 diaromatics 0.01 aliphatics	Condition index reduced

(Continued)

ff Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 1 (Concluded)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Gilfillan and Vandermeulen 1978	Bunker C (Arrow spill)	Bivalve (M)	Field collection 6 years after spill	3800 µg/g sediment	200	1-2 year lag in growth
Thomas 1978	Bunker C (Arrow spill)	Bivalve (M)	Field collection 6 years after spill	5115 µg/g sediment	157	Diminished increase in weight gain per unit length
		Snail (M)			12.2	Diminished increase in weight gain per unit length

Table 2

References in which Reproduction Was Evaluated in Aquatic Animals Contaminated with Environmental Pollutants

Reference	Contaminant	Organism*	Exposure Time	Exposure Concentration**	Tissue Concentration†	Biological Effect
Benoit et al. 1976	Cadmium	Trout (Fw)	156 weeks, 3 generations	0-3.4	0-10†† (range in gill, kidney, liver)	No effect on % hatched
				6.3	No data	100% mortality
Spehar 1976	Cadmium	Flagfish (Fw)	100 days	0-4.1	0-6	No effect on reproduction
				8.1-31	10-35	Reduced number of spawnings/ female and total embryos produced
Westernhagen and Dethlefsen 1975	Cadmium	Flounder eggs (M)	10 days	0-1.0	0-0.06††	% hatched and hatch rate unaffected
				2-5	0.04-0.16††	% hatched and hatch rate reduced
Westernhagen et al. 1975	Cadmium	Garpike eggs (M)	24 days	0-1	0-0.19††	No effect on reproduction
				2-5	0.19-0.36††	% viable hatch increased
Westernhagen et al. 1974	Cadmium	Herring eggs (M)	14 days	0.1-5.0	0.04-0.76††	No effect on hatching rate
Zaroogian and Morrison 1981	Cadmium	Oyster (M)	33-37 weeks	5-15	18-54	Production of abnormal larvae
Marshall 1978	Cadmium	Crustacean (Fw)	22 weeks	1-8	3.5-10.3††	Reduced longevity and increased prenatal mortality partially offset by an increase in brood size and number of ovigerous females; carrying capacity (k) inversely related to tissue concentrations
Hatakeyama and Yasuno 1981	Cadmium	Arthropod (Fw) (Cladocera)	14 days	14.3-71.2 µg/g dry food	200-225††	No effect on number of young produced
				341.3 µg/g dry food	450††	Decreased number of young produced

(Continued)

* Parenthetical entries after name of organism are defined as follows: (Fw) = Freshwater; (M) = Marine.
** Entries for exposure concentration are in units of micrograms per liter (µg/ℓ) unless noted otherwise.
† Entries for tissue concentration are in units of micrograms per gram (µg/g) wet weight whole animal unless noted otherwise.
†† Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 2 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Heisinger and Green 1975	Mercury	Ricefish (Fw)	16 days	0-10	0-16	No effect on hatching
				15	29	Reduced percent hatching
				20-30	54-56	No hatching
Snarski and Olson 1982	Mercury	Fathead minnows (Fw)	41 weeks	0-0.5	0-2.84	No effect on reproduction
				1.02-3.69	4.47-18.8	Spawning completely inhibited
McKim et al. 1976	Mercury	Trout (Fw)	144 weeks, 3 generations	0-0.29	0-3.4	No effect on reproduction and survival
				0.93	9.4	Reduced spawning and percent hatched
				2.93	No data	Very high mortalities
Biesinger et al. 1982	Mercury	Arthropod (Fw) (Cladocera)	21 days	0.36-0.72	8.59-15.26	No effect on number of young produced
				1.28	23.28	Decreased number of young produced
				2.70	No data	100 percent mortality
Westernhagen et al. 1981	Mercury	Flounder (M)	Field collected	No data	0.0007-0.065 (ovaries)	Viability of hatch did not correlate with metal concentration in ovaries
	Zinc				3.7-31.7 (ovaries)	
	Cadmium				0.0004-0.012 (ovaries)	
Pierson 1981	Zinc	Guppy (Fw)	134 days, 1 generation	173-328	0.10-0.26††	No effect on reproduction
				607	0.30††	Slight reduction in number of females producing broods; slight increase in the time to first brood
Holcombe et al. 1979	Zinc	Trout (Fw)	136 weeks, 3 generations	0-265	No data	No effect on reproduction
				534	20-30††	Slight reduction in percent hatched
				1360	No data	Severe reduction in percent hatched
Spehar 1976	Zinc	Flagfish (Fw)	100 days	0-267	0-300	No effect on reproduction

(Continued)

†† Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 2 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Holcombe et al. 1976	Lead	Trout (Fw)	144 weeks, 3 generations	0-58	4-12†† (range in	No effect on reproduction
				119-235	12-60†† liver, gill, and kidney	Reduced spawning and percent hatched
				473	No data	Complete inhibition of spawning and high mortality
Benoit 1975	Copper	Bluegill fish (Fw)	88 weeks, 1 generation	0-21	0.6-2.4†† (range in	No effect on reproduction
				40-77	1.0-11.4†† liver, gill, and kidney)	Reduction in number of eggs/spawn and number of spawns/female
				162	2.6-96††	Spawning completely inhibited
Oshida and Word 1982	Chromium	Worm (M)	300 days, 2 generations	2.6-16.6	0.040-4.42	No effect on reproduction
				38.2	6.03-8.28	Reduced number of offspring by second generation worms
Hansen et al. 1975	PCB (Aroclor 1016)	Sheepshead minnow (M)	29 days	1-10	5.4-110 (adults) 4.2-66 (eggs)	No effect on egg fertility, hatching, or subsequent survival of progeny
				32	200-1100 (adults)	100 percent mortality in adults
Bengtsson 1980	PCB (Clophen A50)	Minnow (Fw)	40 days	125 µg/0.5 g dry food/day/fish	1.3	No effect on reproduction
				135-1250 µg/0.5 g dry food/day/fish	10-100	Reduction in time to hatching with resultant fry dying
Zitko and Sanders 1979	PCB (Aroclor 1254)	Salmon (Fw)	Field collected	No data	0.23-0.42 (eggs)	No correlation between hatching success and tissue concentration
	Hexachlorobenzene				0.006-0.010 (eggs)	
	DDT and metabolites				0.01-0.031 (eggs)	

(Continued)

†† Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water

Table 2 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Westernhagen et al. 1981	PCB	Flounder (M)	Field collected	No data	5000-31,700 (ovaries)	Reduced viable hatch at PCB tissue concentrations above 120,000
	DDD DDE Hexachlorobenzene Dieldrin Heptachlorepoxide				300-30,000 (ovaries) 100-62,000 (ovaries) 60-2000 (ovaries) 100-49,000 (ovaries) 80-3000 (ovaries)	Hatch viability not correlated with tissue concentration of any other contaminant
Goodman et al. 1982	Kepone	Sheepshead minnow (M)	90-133 days	0.041-0.074	0.15-0.56	Increased number of eggs/female/day; fertility unaffected
				0.12-0.39	0.86-3.0	Number of eggs/female/day unaffected; fertility unaffected
				0.78	5.0-6.8	Decreased number of eggs/female/day; reduced fertility
Hansen et al. 1977a	Kepone	Sheepshead minnow (M)	28 days	0.05-0.80	0.26-4.7	Production of normal embryos
				1.9	11	Production of abnormal embryos
Hansen et al. 1977b	Endrin	Sheepshead minnow (M)	23 weeks, 1 generation	0.027-0.12	0.20-1.0 (adults) 0.09-0.87 (eggs)	No effect on reproduction
				0.31	0.94 (adults) 1.80 (eggs)	Reduced fertilization and early hatching; high mortalities
				0.72	No date	
Brown and Thompson 1982	Phthalates	Crustacean (Fw)	21 days, 1 generation	1.33-115	0.32-26.8	No effect on number of progeny produced
Spehar et al. 1983	Permethrin (Synthetic pyrethroid)	Minnows (Fw)	32 days	0.11-0.66	0.19-2.16	Normal larvae produced and percent hatchability unaffected
				1.40	4.51	Abnormal larvae produced and percent hatchability unaffected
	AC 222, 705 (Synthetic pyrethroid)	Minnows (Fw)	32 days	0.02-0.07	0.06-0.17	Normal larvae produced and percent hatchability unaffected

(Continued)

Table 2 (Concluded)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Spehar et al. 1983 (Cont'd)				0.13-0.29	No data	Abnormal larvae produced and percent hatchability unaffected; high mortalities
Goodman et al. '79	Diazinon (organophosphate)	Sheepshead minnow (M)	108 days	0.47-6.50	0.05-2.4	Number of eggs/female/day inversely proportionate to tissue concentrations; egg fertility unaffected
Hannah et al. 1982	Benzo(a) pyrene	Rainbow trout (Fw)	36 days	2.4	12.34	No effect on hatching success or hatching time
Widdows et al. 1982	North sea crude oil	Mussel (M)	140 days	30	152.1 aromatics (digestive gland) 22.9 aromatics (remaining tissues)	No effect on the production and maturation of ripe gemetes

Table 3

References in Which Morphology/Histology Was Evaluated in Aquatic Animals Contamined with Environmental Pollutants

Reference	Contaminant	Organism*	Exposure Time	Exposure Concentration**	Tissue Concentration†	Biological Effect
Muramoto 1980	Cadmium	Carp fish (Fw)	90 days	50	9.6†† (viscera) 2.7†† (gills) 0.43†† (other)	Extensive damage to vertebrae
Muramoto 1981	Cadmium	Carp fish (Fw)	100 days	10-100	0.48-1.8†† (viscera) 0.19-0.25†† (gills) 0.004-0.007†† (vertebrae)	Number of deformed vertebrae inversely proportional to cadmium concentration
Beattie and Pascoe 1978	Cadmium	Trout eggs (Fw)	100 hr	28-7700	1.16-114 (eggs) 0.21-1.7 (fry)	Deformed vertebrae and blood clots in fins
				3940	No data	100 percent mortality
		Trout fry (Fw)	320 hr	28	0.71 (fry)	Deformed vertebrae and blood clots in fins; 100 percent mortality
				170-3940	No data	
Carmichael and Fowler 1981	Cadmium	Bivalve (M)	5 days	700	200 (kidney)	Tissue turned from normal dark brown to light brown by concentrations formed in kidneys
Westernhagen et al. 1974	Cadmium	Herring eggs (M)	14 days	0.1-5.0	0.04-0.76‡	Yolk sac volume directly proportional to concentration probably due to decreased activity; length of fry at hatching inversely proportional to cadmium concentration
Westernhagen et al. 1975	Cadmium	Garpike eggs (M)	24 days	0-0.5	0-0.12‡	No effect on heartbeat and normal pectoral fin movement
				1-5	0.12-0.36‡	Diminished heartbeat and normal pectoral fin movement
Westernhagen and Dethlefsen 1975	Cadmium	Flounder eggs (M)	10 days	0-5	0-0.16‡	Length of fry at hatch, eye, and otic capsule diameter unaffected

(Continued)

* Parenthetical entries after name of organism are defined as follows: (Fw) = Freshwater; (M) = Marine.
** Entries for exposure concentration are in units of micrograms per liter (µg/ℓ) unless noted otherwise.
† Entries for tissue concentration are in units of micrograms per gram (µg/g) wet weight whole animal unless noted otherwise.
†† Data originally reported on an ash basis were converted to wet weight assuming 80 percent body water and with percent ash values reported by authors.
‡ Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 3 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Heisinger and Green 1975	Mercury	Ricefish eggs (Fw)	16 days	0-10	0-16	No effects observed
				15-30	29-56	Subcutaneous hemorrhaging; deterioration and reduction of blood vessels; hemolysis of red blood cells; deformed vertebral column
Snarski and Olson 1982	Mercury	Flathead minnows (Fw)	41 weeks	0-1.02	0-4.47	No effect on vertebral column
				2.01-3.69	9.41-18.8	Deformed vertebral column
Wobeser 1975	Mercury	Trout (Fw)	105 days	0-24,000	0-27 (muscle)	Gill hyperplasia
Kendall 1975	Mercury	Catfish (Fw)	Single injection	15,000	14.2 (kidney after 96 hr)	Loss of distinctive features in glomeruli
Kendall 1977	Mercury	Catfish (Fw)	Single injection	15,000	157 (liver after 96 hr)	Sloughing of exterior cells in liver and kidney; exocrine pancreatic tissue disintegrated; liver parenchyma tissue unaffected
Weis and Weis 1982	Mercury	Killifish eggs (M)	7 days	50	5.4	Minimal effect on eye, heart, muscle, and bone development
					8.4	Maximal effect on eye, heart, muscle, and bone development
Holcombe et al. 1976	Lead	Trout (Fw)	144 weeks	0-58	4-12‡	No effect on vertebral column
				119-235	12-60‡	Deformed vertebral column
Cardeilhac et al. 1979	Copper	Fish (M)	17 hr	8500	0.1-1.0 (gills)	Gill lamellae blunt with dilated mucous cells
					4.3-16.3 (liver)	Liver unaffected; kidneys and kidney capillaries swollen and congested
Young et al. 1981	Copper	Worm (M)	5-10 days	10	25-50	Shortening of pinnules on branchial crown
			2-3 days	15-20	50	Shortening of pinnules on branchial crown; swollen mitochondria; disorganized microville; extensive tissue necrosis
			2-3 days	40	50	High mortalities

(Continued)

‡ Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 3 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Sorensen et al. 1982	Selenium	Redear sunfish (Fw)	Field collected	No data	2.80 (hepatopancreas)	No effect
					8.38-11.03 (hepatopancreas)	Degenerative follicles in ovaries; hypoxic vacuolization in veins
		Green sunfish (Fw)	Field collected	No data	1.31 (hepatopancreas)	No effect
					6.05-9.30 (hepatopancreas)	Degenerative follicles in ovaries; hypoxic vacuolization in veins; thickened and vacuolated gills with inflammatory cells
Mehrle et al. 1982	PCB, lead, cadmium, arsenic, selenium, and zinc	Striped bass (Fw)	Field collected	No data	PCB 0.03-0.08; Cadmium 0.05; Lead 0.46-0.80	Strongest vertebrae in fish from "clean" environment
					PCB 0.31-1.44; Cadmium 0.05-0.26; Lead 1.23-1.65	Weakest vertebrae in fish from "polluted" environment
					Arsenic 0.06-0.2; Selenium 0.08-0.57; Zinc 35.7-48.3	No significant variations at different locations. Arsenic, selenium, and zinc concentrations did not correlate with bone strength
Sangalang et al. 1981	PCB (Aroclor 1254)	Fish (M)	5-1/2 months	1-50 µg/g food	0.06-5.3 (testes)	Response intensified as tissue concentration increased and progressed from testicular fibrosis to inhibition of spermatogenesis and finally to complete disintegration of the testes
Freeman et al. 1982	PCB (Aroclor 1254)	Codfish (M)	5-1/2 months	1-50 µg/g wet food	0.02-0.98 (muscle)	Hyperplasia of gills with disrupted blood spaces
					0.04-2.1 (head kidney)	No effect on histopathology of head kidney
					0.06-5.3 (testes)	Hyperplasia and thickening of testes with some necrosis
					10.1-374 (liver)	Degeneration of liver's fatty tissue
Couch and Courtney 1977	PCB (Aroclor 1254)	Shrimp (M)	35 days	0.6-0.7	2 (muscle) 21 (hepatopancreas)	Increased occurrence of the pathogen Baculovirus

(Continued)

Table 7 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Fisher 1980	Kepone	Crab (M)	65 days	0-0.42-2.50 µg/g food	0.38-4.61	Carapace thickness-to-width ratio inversely related to concentration
Hansen et al. 1977a	Kepone	Killifish (M)	28 days	0-1.9	0.26-11	Response intensified as tissue concentration increased. Response progressed from deformed vertebral column, hemorrhaging near brain, and darkened posterior to increased hemorrhaging and fin rot
Mayer et al. 1978	Toxaphene	Catfish (Fw)	5 months	0-0.475	4.0-9.4	Spinal deformities and skin lesions inversely related to concentration
Emanuelsen et al. 1978	Dieldrin	Oyster (M)	43 days	1-100	25.6-2685‡	No effect on fibrous or cellular components of gills, gut, or mantle; no inflammation or infiltration of leucocytes
Poels et al. 1980	Rhine River water	Trout (Fw)	18 months	No data	1.6-PCB 0.12-DDE 0.11-Dieldrin 2.5-PCP	Decreased number of lymphoid cells in spleen; increased number of macrophages with iron inclusions in the spleen
Wells and Cowan 1982	Trifluralin (fluorinated herbicide)	Salmon (Fw)	11 hr	500	100	Compression and fusion of vertebrae
Goodman et al. 1979	Diazinon (organophosphate)	Killifish (M)	108 days	0-6.5	0-2.4	Darkened body due to hemorrhaging and abnormal anterior projection of pectoral fins increased at higher concentrations
Hannah et al. 1982	Benzo(a) pyrene	Rainbow trout (Fw)	36 days	2.4	12.34	Lack of pigmentation, insufficient yolk sacs, cyclopia, shortened body, incomplete development of external body parts
Woodward et al. 1981	Wyoming crude oil	Trout (Fw)	90 days	100-150	14-19	Caudal fin erosion
				450-520	20-30	Caudal fin erosion and gill and eye lens lesions

(Continued)

‡ Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 3 (Concluded)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
McCain et al. 1978	North slope crude oil	Flatfish (M)	4 months	400 µg/g dry sediment	0.037 after 51 days (liver) (accumulation of mono and diaromatics)	Increase in lipid droplets, vacuoles and endoplasmic reticullum in liver; no effect on spleen, kidney, gills, intestine, skin, or fins; emaciated appearance
Widdows et al. 1982	North sea crude oil	Mussel (M)	140 days	30	152.1 aromatics (digestive gland)	Decrease in epithelial cell size
					22.9 aromatics (remaining tissue)	No effect on development of gametic tissues

Table 4

References in Which Behavior Was Evaluated in Aquatic Animals Contaminated with Environmental Pollutants

Reference	Contaminant	Organism*	Exposure Time	Exposure Concentration**	Tissue Concentration†	Biological Effect
Beattie and Pascoe 1978	Cadmium	Trout eggs (Fw)	100 hr	28-7700	1.16-114†† (eggs) 0.21-1.7†† (hatched fry)	Erratic swimming behavior linked to deformed vertebrae
				3940	No data	100 percent mortality
		Trout fry (Fw)	320 hr	28	0.71†† (fry)	Erratic swimming behavior linked to deformed vertebrae
				170-3940	No data	100 percent mortality
Westernhagen et al. 1974	Cadmium	Herring eggs (M)	14 days	0.1-5.0	0.04-0.76††	At increased tissue concentrations, decrease in "normal" rotation of embryos in egg and increase in "abnormal" trembling
Westernhagen et al. 1975	Cadmium	Garpike eggs (M)	24 days	0-0.5	0-0.12††	No effect on behavior
				1.0-5.0	0.12-0.36††	Diminished heartbeat and abnormal movement of pectoral fins
Rogers and Beamish 1982	Mercury	Trout (Fw)	3 months	24-95 µg/g food	11-30	Appetite inversely related to concentration; lethargic behavior and dark coloration
Barthalmus 1977	Mercury	Grass shrimp (M)	30 days	50	1.1-2.1	Decreased sensitivity to negative reinforcement (electrical shock)
Somero et al. 1977a,b	Lead	Fish (M)	87 days	2650 µg/kg seawater	80†† (gills) 3†† (muscle) 200†† (spleen)	Hyperactive behavior
Cardeilhac et al. 1979	Copper	Fish (M)	12-17 hr	8500	0.10 (gill) 4.3 (liver)	Lethargic - correct posture and some avoidance of capture
					0.14 (gill) 9.4 (liver)	Indifference - correct posture but no avoidance of capture
					1.00 (gill) 16.3 (liver)	Incoordination - partial loss of normal posture and disoriented movement; no avoidance of capture

(Continued)

* Parenthetical entries after name of organism are defined as follows: (Fw) = Freshwater; (M) = Marine.
** Entries for exposure concentration are in units of micrograms per liter (µg/ℓ) unless noted otherwise.
† Entries for tissue concentration are in units of micrograms per gram (µg/g) wet weight whole animal unless noted otherwise.
†† Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 4 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Young et al. 1981	Copper	Worm (M)	5-10 days	0-20	25-50	No abnormal behavior
			2-3 days	40	50	Left burrows, came to surface, and died
Olofsson and Lindahl 1979	Mercury PCB DDT	Fish (M)	Field collected	No data	150 110 7	Decreased ability to maintain position while swimming in a current
Anonymous 1981	PCB and DDE	Trout (Fw)	6 months	10 ng PCB/ℓ + 1.0 ng DDE/ℓ + 1.0 µg PCB/g food + 0.1 µg DDE/g food	1.53 PCB + 1.0 DDT + DDE	No effect on swimming performance, predator avoidance or temperature preference
				5X the above dose	5.06 PCB + 2.4 DDT + DDE	No effect on swimming performance, predator avoidance, or temperature preference
				25X the above dose	26.3 PCB + 9.95 DDT + DDE	No effect on swimming performance or predator avoidance; preference for lower temperatures exhibited
Bengtsson 1980	PCB	Minnows (Fw)	130 days	25-2500 µg/g dry food	0-100	No effect on swimming performance
Tagatz 1976	Mirex	Grass shrimp (M)	14-16 days	0.011-0.130	0.02-0.20	Diminished ability to avoid predation
Tagatz et al. 1976	Mirex	Oystem (M)	10 weeks	0.038	1.3-28	Diminished ability to withstand predation
		Mussel (M)	10 weeks	0.038	1.6-2.0	Dimished ability to withstand predation
Fisher 1980	Kepone	Blue crab (M)	65 days	0.38-1.64 µg/g food	0.38-1.73	No effect on behavior
				2.26-2.50 µg/g food	2.54-4.61	Excitable behavior during feeding; reduced ability to locate and consume food
Hansen et al. 1977a	Kepone	Sheepshead minnow (M)	28 days	0-1.9	0.26-11	Erratic swimming behavior and a reduction in feeding rate both increased as concentrations increased
Goodman et al. 1979	Diazinon (organophosphate)	Sheepshead minnow (M)	108 days	0.47-6.5	0.05-2.40	Abnormal anterior projection of pectoral fins

(Continued)

Table 4 (Concluded)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Folmar and Hodgins 1982	No. 2 fuel oil	Salmon (M)	17 days	~800	3581 µg/kg (liver)	Diminished feeding; lethargic behavior
	PCB (Aroclor 1254)		Single injection	150 µg/kg	329 µg/kg (liver)	
	PCB + No. 2 fuel oil		Combination of above	Combination of above	309 µg PCB + 1411 g hydrocarbons/kg (liver) (accumulation of naphthalenic aromatics)	
Anderson 1975	No. 2 fuel oil	Killifish (M)	4 hr	6280 1930 TN‡	25-66,704 TN‡ (range in various tissues)	Progressively severe swimming behavior observed as tissue concentrations (especially brain tissue) increased
Krebs and Burns 1977	No. 2 fuel oil	Fiddler crab (M)	7 years after spill	200-10,000 µg/g sediment	280	Shallow-burrow construction; lethargic behavior; increased molting; display of courtship colors during non-reproductive season
Tatem 1977	No. 2 fuel oil	Grass shrimp (M)	8 hr	2600 550 TN‡	33 TN‡	Hyperactive and highly excitable
Stekoll et al. 1980	Prudhoe Bay crude oil	Bivalve (M)	6 months	30	200	Burrowing rate not affected
Clement et al. 1980	Prudhoe Bay crude oil	Bivalve (M)	6 months	300-3000	700-1000	Reduced burrowing rate
Roesijadi and Anderson 1979	Prudhoe Bay crude oil	Bivalve (M)	55 days	1233 µg/g sediments	0.14-0.42 aliphatics 1.15-5.21 naphthalenes	Abnormal surfacing from burial in sediments
McCain et al. 1978	North Slope crude oil	Flatfish (M)	4 months	400 µg/g sediments	0.037 †† after 51 days (liver) (mostly mono diaromatics)	Diminished feeding rates; lethargic behavior

‡ TN = total napthalenes.

Table 5

References in Which Metabolism Was Evaluated in Aquatic Animals Contaminated with Environmental Pollutants

Reference	Contaminant	Organism*	Exposure Time	Exposure Concentration**	Tissue Concentration†	Biological Effect
Dickson et al. 1982	Cadmium	Crayfish (Fw)	21 days	3.3-9.4	0.21-1.42†† (muscle)	No effect on muscle oxygen consumption
					36-41†† (gills)	No effect on gill oxygen consumption
Vernberg et al. 1977	Cadmium	Grass shrimp (M)	7 days	50	2-18	Highly variable oxygen consumption; no correlation with cadmium concentration
Poulsen et al. 1982	Cadmium	Mussel (M)	17 days	8.4-109	2-30††	No effect on net metabolism and its inherent components, i.e., oxygen consumption, ingestion, or assimilation rates
Calabrese et al. 1975	Cadmium	Flounder (M)	60 days	5-10	<0.3 (gills)	No effect on oxygen consumption
	Mercury	Flounder (M)	60 days	5	21 (gills)	No effect on oxygen consumption
				10	43 (gills)	Increased oxygen consumption
Somero et al. 1977a,b	Lead	Fish (M)	87 days	2650 µg/kg seawater	80†† (gills) 3†† (muscle) 200†† (spleen)	Gill oxygen consumption not affected but whole animal oxygen consumption elevated
Anderson 1978	Lead	Crayfish (Fw)	40 days	0-2000	1-6††	No effect on oxygen consumption
Cardeilhac et al. 1979	Copper	Fish (M)	12-17 hr	8500	0.10 (gill) 4.3 (liver)	No effect on oxygen consumption
					0.14 (gill) 9.4 (liver)	Increased oxygen consumption
					1.0 (gill) 16.3 (liver)	No effect on oxygen consumption
Wilson 1983	Nickel	Bivalve (M)	14-16 days	0.1-10.0	253-1974††	No effect on oxygen consumption
				100	No data	No effect on oxygen consumption but high mortalities

(Continued

* Parenthetical entries after name of organism are defined as follows: (Fw) = Freshwater; (M) = Marine.
** Entries for exposure concentration are in units of micrograms per liter (µg/ℓ) unless noted otherwise.
† Entries for tissue concentration are in units of micrograms per gram (µg/g) wet weight whole animal unless noted otherwise.
†† Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 5 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Neff and Giam 1977	PCB (Aroclor 1016)	Horseshoe crab (M)	96 days	0.35-71.5	0.08-92.8	No ecologically significant change in oxygen consumption; highly variable oxygen consumption
	Halowax 1099 (chlorinated naphthalene)	Horseshoe crab (M)	96 days	22-70	0.51-5.7	
Anonymous 1981	PCB and DDE	Trout (Fw)	6 months	10 ng PCB/ℓ + 1.0 ng DDE/ℓ + 1 µg PCB/g food + 0.1 µg DDE/g food	1.53 PCB + 1.0 DDT + DDE	No effect on oxygen consumption
				5X the above dose	5.06 PCB + 2.4 DDT + DDE	
				25X the above dose	26.3 PCB + 9.95 DDT + DDE	
Fisher 1980	Kepone	Blue crab (M)	65 days	0-1.6 µg/g food	0-1.73	No effect on oxygen consumption
				2.50 µg/g food	4.61	Elevated rates of oxygen consumption
Ginkel 1981	Atrazine	Whitefish (M)	25-30 days	300	0.48	Increased routine oxygen consumption
				600 µg/kg food	0.25	Increased routine oxygen consumption
Stainken 1976	No. 2 fuel oil	Bivalve (M)	4 weeks	4500	20-30	Elevated rates of oxygen consumption
Stainken 1978	No. 2 fuel oil	Bivalve (M)	4 weeks	43,700-60,700	60-145	No effect on oxygen consumption
Tatem 1977	No. 2 fuel oil	Grass shrimp (M)	5 hr	3000-3600 600 TN‡	1.8-2.1 TN‡	Reduction in oxygen consumption
Gilfillan et al. 1976	No. 6 fuel oil (Tamano spill)	Bivalve (M)	1 year after spill	11,760 µg/g sediments	66.1	Elevated rates of oxygen consumption
Widdows et al. 1982	North sea crude oil	Mussel (M)	28 days	36	21.8-78.3 aromatics (digestive gland) 8.8-16.2 aromatics (remaining tissues)	Decreased scope for growth and food absorption efficiency; elevated oxygen consumption rates

(Continued)

‡ TN = total napthalenes.

Table 5 (Concluded)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Widdows et al. 1982 (Cont'd)			140 days	30	152.1 aromatics (digestive gland) 22.9 aromatics (remaining tissues)	Decreased scope for growth and food absorption efficiency; no effect on oxygen consumption; increased ammonia excretion
Clement et al. 1980	Prudhoe Bay crude oil	Bivalve (M)	180 days	0-30	0-200	No effect on oxygen consumption rates
Stekoll et al. 1980	Prudhoe Bay crude oil	Bivalve (M)	180 days	300-3000	700-1000	Reduced oxygen consumption rates
Levitan and Taylor 1979	Naphthalene	Killifish (M)	12 hr	4000	12-23	Elevated oxygen consumption rates
Dillon 1983	Dimethylnaphthalene	Grass shrimp (M)	32 days	0.24 µg/g wet food	4.12-4.32	No effect on oxygen consumption at normoxic concentrations; elevated oxygen concentrations decreased

Table 6

References in Which Osmotic/Ionic Regulation Was Evaluated in Aquatic Animals Contaminated with Environmental Pollutants

Reference	Contaminant	Organism*	Exposure Time	Exposure Concentration**	Tissue Concentration†	Biological Effect
Calabrese et al. 1975	Cadmium	Flounder (M)	60 days	5-10	0.3 (gills)	No effect on plasma osmolality
	Mercury	Flounder (M)	60 days	5	21 (gills)	Decreased plasma osmolality
				10	43 (gills)	No effect on plasma osmolality
Dillon and Anderson 1979; Dillon and Neff 1978	Mercury	Clam (M)	14 days	50	40	No effect on ultimate concentrations of Na^+, K^+, nitrogenous compounds or total osmotic pressure when transferred to reduced salinity; slower adaptability to reduced salinity by exposed clams
Cardeilhac et al. 1979	Copper	Fish (M)	12-17 hr	8500	0.1-1 (gill 4.3-16.3 (liver)	Increased serum K^+
Roesijadi 1980	Copper	Bivalve (M)	30 days	0-18	0-21.8†† (gill)	Regulation of Na^+ and K^+ unaffected; high survival
				39-82	30†† (gill)	Regulation of Na^+ and K^+ disrupted; low survival
Thomas et al. 1981	Pentachlorophenol	Fish (M)	5 days	100	37.1	Reduction in total osmotic pressure
Caldwell 1974	Methoxychlor	Crab (M)	7 days	10	0.31 2.0 (gill)	No effect on total osmotic pressure
		Crab (M)	14 days	10	1.0 2.5 (gill)	No effect on total osmotic pressure, Na^+ or K^+ regulation but Ma^{++} regulation disrupted
Thomas et al. 1980	No. 2 fuel oil	Fish (M)	3 hr	200-300 TN‡	300 TN‡ (liver) 150 TN‡ (brain) 100 TN‡ (muscle)	Increased total osmotic pressure
			12-14 hr	100 TN‡	100 TN‡ (liver) 600 TN‡ (brain) 10 TN‡ (muscle)	Return of total osmotic pressure to control levels

* Parenthetical entries after name of organism are defined as follows: (Fw) = Freshwater; (M) = Marine.
** Entries for exposure concentration are in units of micrograms per liter (µg/ℓ) unless noted otherwise.
† Entries for tissue concentration are in units of micrograms per gram (µg/g) wet weight whole animal unless noted otherwise.
†† Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.
‡ TN = total naphtalenes.

Table 7

References in Which Enzymes Were Evaluated in Aquatic Animals Contaminated with Environmental Pollutants

Reference	Contaminant	Organism*	Exposure Time	Exposure Concentration**	Tissue Concentration†	Biological Effect
Pruell and Engelhardt 1980	Cadmium	Killifish (M)	4 days	1000	4 (liver)	Liver catalase unaffected
				10,000-25,000	10-42 (liver)	Liver catalase inhibited
Shore et al. 1975	Cadmium	Mollusc (M)	Field collected	No data	5.4-51.4††	Activity of glycolytic enzymes unaffected
					83.8-107††	Activity of glycolytic enzymes diminished
Manen et al. 1976	Mercury	Flounder (M)	Single injection	2 mg/kg	13-33 in 5-24 hr	Decreased ornithine decarboxylase activity
					3-6 in 48-72 hr	Increased orithine decarboxylase activity
Lockhart et al. 1972	Mercury	Fish (Fw)	Field collected	No data	2.7	No effect on enzymes
					7-8	Decrease in alkaline phosphatase in blood
Haya et al. 1983	Zinc	Lobster (M)	4 days	25,000-60,000	2570 (gills)	Decreased Na^+, K^+ ATPase activity in the gills
Hodson et al. 1978b	Lead	Trout (Fw)	36 hr	100	40 µg/ℓ (blood)	Decreased activity of delta-amino levulinic acid dehydrogenase activity in red blood cells
Hodson et al. 1980	Lead	Trout (Fw)	4 months	32-100	0.3-0.6 µg/ml (blood)	Decreased activity of delta-amino levulinic acid dehydrogenase in red blood cells
Hodson et al. 1978a	Lead	Trout (Fw)	8 months	13	2.0 µg/ml (blood) 10 (opercular bone)	Decreased activity of delta-amino levulinic acid dehydrogenase in red blood cells
Roesijadi 1980	Copper	Bivalve (M)	30 days	0-39	0-30†† (gill)	Increased acid phosphatase activity in the gill

(Continued)

* Parenthetical entries after name of organism are defined as follows: (Fw) = Freshwater; (M) = Marine.
** Entries for exposure concentration are in units of micrograms per liter (µg/ℓ) unless noted otherwise.
† Entries for tissue concentration are in units of micrograms per gram (µg/g) wet weight whole animal unless noted otherwise.
†† Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 7 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Viarengo et al. 1981b	Copper	Mussel (M)	3 days	40	10 (digestive gland)	Lysosome stability reduced, i.e., reduced latency of hexosaminidase in digestive gland cells
Harrison and Berger 1982	Copper	Mussel (M)	3-4 weeks	0-75	0-8†† (digestive gland)	Lysosome stability inversely related to concentration, i.e., reduced latency of hexosaminidase in digestive gland cells
Kuhnert and Kuhnert 1976	Chromium	Trout (Fw)	2 days	2500	2.164 (kidney)	Decreased Na^+, K^+ ATPase in kidney; no effect on MG^{++} ATPase
					2.141 (gill) 0.579 (intestine) 0.544 (liver)	No effect on Na^+, K^+ ATPase, or Mg^{++} ATPase in gill, intestine, or liver
Addison et al. 1978	PCB (Aroclor 1254)	Trout (Fw)	18 days	1.65 mg/fish/feeding (7 feedings total)	39	Increased ethoxycoumarin O-demethylase activity; no effect on the activities of aniline hydroxylase, or cytochrome P-450
Neufeld and Pritchard 1979	DDT	Crab (M)	50 hr	Single injection of 100 µg/kg	0.06 (gill) 1.5 (hepatopancreas) 0.04 (blood)	Decreased NA^+, K^+ ATPase activity in gill
Vink 1975	Dieldrin	Flatfish (M)	23-30 days	0.4	5.0 (liver) 1.2 (muscle)	Decreased aminopyrine demethylase activity in the liver; no effect on aniline hydroxylase activity in the liver
Mayer et al. 1978	Toxaphene	Catfish (Fw)	5 months	0-0.475	4.0-9.4	Increased aromatic hydrocarbon hydroxylase activity
Goodman et al. 1979	Diazinon (organophosphate)	Sheepshead minnow (M)	108 days	0-6.5	0-2400	Acetylcholinesterase activity in the brain inversely related to concentration
Burns 1976; Krebs and Burns 1977	No. 2 fuel oil	Fiddler crab (M)	4 years after spill	200-10,000 µg/g sediment	180-280	No effect on the enzymes of the mixed function oxidase (MFO) system

(Continued)

Table 7 (Concluded)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Clement et al. 1980; Stekoll et al. 1980	Prudhoe Bay crude oil	Bivalve (M)	6 months	0-3000	200-1000	No effect on Na^{+}, or K^{+} ATPase, or Mg^{++} ATPase activities
Widdows et al. 1982	North Sea crude oil	Mussel (M)	140 days	30	152.1 aromatics (digestive gland) 22.9 aromatics (remaining tissues)	Lysosome stability reduced, i.e., reduced latency of hexosaminidase in digestive gland cells; increased activities of phosphofructokinase, NADP - isocitrate dehydrogenase, phosphoenolpyruvate carboxylase, and glucose - 6 - dehydrogenase; no effect on the activities of hexokinase or pyruvate kinase
Dillon and Fisher 1983; Dillon 1982	Dimethylnaphthalene	Grass shrimp (M)	32 days	0.24 µg/g food	5.26-7.20	No effect on acid phosphatase activity in the blood

Table 8

References in Which Biochemical Compositon Was Evaluated in Aquatic Animals Contaminated with Environmental Pollutants

Reference	Contaminant	Organism*	Exposure Time	Exposure Concentration**	Tissue Concentration†	Biological Effect
Thomas et al. 1982	Cadmium	Fish (M)	42 days	10,000	240†† (liver)	Liver ascorbic acid decreased by 60 percent
					70†† (gill)	Gill ascorbic acid decreased by 50 percent
					16†† (kidney)	Kidney ascorbic acid decreased by 60 percent
					1.4†† (brain)	Brain ascorbic acid decreased by 18 percent
Dickson et al. 1982	Cadmium	Crayfish (Fw)	21 days	0-9.4	0-1.42†† (muscle) 0-41†† (gills)	No effect on total adenylates, ATP turnover, or adenylate energy charge ratio in muscle or gill tissue
Carr and Neff 1982	Cadmium	Worm (M)	7 days	40,000	110††	No effect on ascorbic acid, coelomic fluid glucose, or total glycogen after 7 days
			55 days	10,000	280††	No effect on coelomic fluid glucose but total glycogen reduced after 55 days; increase in ascorbic acid in the tissues after 55 days
Haya et al. 1983	Zinc	Lobster (M)	4 days	25,000-60,000	2570 (gills)	No effect on adenylate energy charge in gills, hepato-pancreas, or tail muscle
Kearns and Atchison 1979	Cadmium	Fish (Fw)	Field collected	No data	0.22-0.57††	RNA:DNA ratios directly related to cadmium concentration
	Zinc	Fish (Fw)	Field collected	No data	25-32††	No relationship between RNA: DNA ratios and zinc concentration
Calamari et al. 1982; Arillo et al. 1982	Cadmium Chromium Nickel	Trout (Fw)	6 months	10 200 1000	0.42 (liver) 2.18 (liver) 2.18 (liver)	No effect on protein content in the liver; decrease in liver glucides and gill sialic acid

(Continued)

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** Entries for exposure concentration are in units of micrograms per liter (µg/ℓ) unless noted otherwise.
† Entries for tissue concentration are in units of micrograms per gram (µg/g) wet weight whole animal unless noted otherwise.
†† Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 8 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Viarengo et al. 1981a	Copper	Mussel (M)	7 days	80	40.9 (gills) 11.2 (digestive gland) 2.64 (mantle)	Decrease in uptake of amino acids, protein synthesis, and ATP content in the gills, digestive gland, and mantle
Viarengo et al. 1982	Copper	Mussel (M)	Field collected from clean and polluted waters	No data	(digestive gland) 0.88 clean 4.64 polluted	Decrease in the uptake of free amino acids and the rate of RNA and protein synthesis in the digestive gland of mussels from polluted environments; copper and mercury, but not zinc and cadmium, elevated in the digestive gland of mussels from polluted water
	Mercury				0.018 clean 0.080 polluted	
	Zinc				19.5 clean 20.8 polluted	
	Cadmium				0.22 clean 0.28 polluted	
Hodson et al. 1980	Lead	Trout (Fw)	4 months	32-100	0.3-0.6 µg/m (blood)	Minimal effect on ascorbic acid content of blood, liver, kidney, brain, or carcass; effects of lead not ameliorated by dietary supplements of ascorbic acid
Sorensen et al. 1982	Selenium	Redear sunfish (Fw)	Field collected	No data	2.80-11.03 (hepatopancreas)	No effect on condition index or hepatopancreas index
		Green sunfish (Fw)	Field collected	No data	1.3 (hepatopancreas)	No effect on condition index or hepatopancreas index
					6.05-9.30 (hepatopancreas)	Decreased hepatopancreas index
Freeman et al. 1982	PCB (Aroclor 1254)	Codfish (M)	5-1/2 months	1-50 µg/g wet food	0.06-5.3 (testes)	Disruption in production of sex steroids from testes
					0.04-2.1 (head kidney) 0.02-0.98 (mussel) 10.1-374 (liver)	Disruption in production of adrenal hormones from head kidney
Leatherland et al. 1979	PCB	Salmon (Fw)	3 months	50 mg/kg food	43	Hepatosomatic index unchanged; liver lipids increased; carcass lipids unchanged
				500 mg/kg food	135	Hepatosomatic index increased; liver lipids increased; carcass lipids decreased

(Continued)

Table 8 (Continued)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Leatherland et al. 1979 (Cont'd)	Mirex	Salmon (Fw)	3 months	5 mg/kg food	1.6	Hepatosomatic index decreased; liver lipids unchanged; carcass lipids unchanged
				50 mg/kg food	9.6	Hepatosomatic index decreased; liver lipids unchanged; carcass lipids decreased
	PCB + Mirex			50 mg PCB/kg food plus 5 mg Mirex/kg food	9.8 PCB 1.9 Mirex	Hepatosomatic index increased; liver lipids unchanged; carcass lipids unchanged
Carr and Neff 1981	Pentachlorophenol	Worm (M)	66 days	100	112	Coelomic glucose increased while tissue glycogen decreased; no effect on ascorbic acid content
Thomas et al. 1981	Pentachlorophenol	Fish (M)	5 days	100	37.1	Plasmaglucose increased while liver glycogen decreased
Mayer et al. 1978	Toxaphene	Catfish (Fw)	5 months	0-0.475	4.0-9.4	Ascorbic acid and collagen content in vertebrae inversely related to concentration; toxic effects of toxaphene ameliorated by dietary supplement of ascorbic acid
Poels and Strik 1975	Rhine River water	Rainbow trout (Fw)	6 months	No data	PCB - 0.12 (liver) Dieldrin - not detectable (liver) Hexachlorobenzene - 0.14 (liver) Pentachlorobenzene - 0.04 (liver)	Increased liver and kidney somatic index
Widdows et al. 1982	North Sea crude oil	Mussel (M)	140 days	30	152.1 aromatics (digestive gland) 22.9 aromatics (remaining tissues)	No effect on free amino acid composition
Augenfield et al. 1980	Prudhoe Bay crude oil	Bivalve (M)	54 days field exposure	850-1237 µg/g sediment	1.77 di and tri-aromatics 0.337 saturates	Decrease in total free amino acids; specific decreases in alanine, lysine, taurine, aspartic, and glutamic acid

(Continued)

Table 8 (Concluded)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Roesijadi and Anderson 1979	Prudhoe Bay crude oil	Bivalve (M)	38 days in the field	364-1144 µg/g sediments	0.46 diaromatics 0.01 aliphatics	Decrease in total free amino acids; specific decrease in alanine, lysine, glycine and threonine
Brannon and Rao 1979	Barite	Grass shrimp (M)	106 days	500,000	900 (abdominal muscle)	Decreased calcium in muscle
					8000 (hepatopancreas)	Decreased calcium in hepatopancreas
					8000 (exoskeleton)	Increased calcium in exoskeleton

Table 9

References in Which Blood Chemistry Was Evaluated in Aquatic Animals Contaminated with Environmental Pollutan.

Reference	Contaminant	Organism*	Exposure Time	Exposure Concentration**	Tissue Concentration†	Bi.ogical Effect
Shore et al. 1975	Cadmium	Mollusc (M)	Field collected	No data	5.4-51.4††	No effect a blood glucose
					83.8-107††	Increase in blood glucose
Calabrese et al. 1975	Cadmium	Flounder (M)	60 days	5-10	0.3 (gills)	No effect on serum hematocrit, hemoglobin, or total protein
	Mercury	Flounder (M)	60 days	5-10	21-43 (gills)	Decreased serum hematocrit and hemoglobin; increased total protein
Lockhart et al. 1972	Mercury	Fish (Fw)	Field collected	No data	2.7	No effect on blood chemistry
					7-8	Some degree of starvation implied by emaciated condition and decrease in serum glucose, proteins, alkaline phosphatase, and cortisol
Hodson et al. 1978a	Lead	Trout (Fw)	8 months	13	2 µg/ml (blood)	Decrease in the volume and iron content of red blood cells offset by increased production of red blood cells
Sorensen et al. 1982	Selenium	Redear sunfish (Fw)	Field collected	No data	2.80-11.03 (heptopancreas)	No effect on hematocrit
		Green sunfish (Fw)	Field collected	No data	1.31 (heptopancreas)	No effect on hematocrit
					6.05-9.30 (heptopancreas)	Decreased hematocrit
Thomas et al. 1981	Pentachlorophenol	Fish (M)	5 days	100	37.1	Increased blood cortisol; no effect on blood cholesterol
Poels et al. 1980	PCB	Trout (Fw)	18 months	No data	1.6 - PCB	Increase in serum glucose and decrease in serum hemoglobin
	Pentachlorophenol				2.5 - PCP	
	DDE				0.12 - DDE	
	Dieldrin				0.11 - Dieldrin	

(Continued)

* Parenthetical entries after name of organism are defined as follows: (Fw) = Freshwater; (M) = Marine.
** Entries for exposure concentration are in units of micrograms per liter (µg/ℓ) unless noted otherwise.
† Entries for tissue concentration are in units of micrograms per gram (µg/g) wet weight whole animal unless noted otherwise.
†† Data originally reported on a dry-weight basis were converted to wet weight assuming 80 percent body water.

Table 9 (Concluded)

Reference	Contaminant	Organism	Exposure Time	Exposure Concentration	Tissue Concentration	Biological Effect
Leatherland and Sonstegard 1980	PCB	Trout (Fw)	1 month	50 mg/kg food	8.05	No effect on triiodothyronine or thyroxine
				500 mg/kg food	40.3	Decreased triiodothyronine and thyroxine
	Mirex			5-50 mg/kg food	0.77-3.51	No effect on triiodothyronine or thyroxine
				500 mg/kg food	4.73	Decreased triiodothyronine; no effect on thyroxine
	PCB + Mirex			50 mg PCB/kg food + 5 mg Mirex/kg food	0.95 PCB 4.85 Mirex	No effect on triiodothyronine or thyroxine
Poels and Strik 1975	Rhine River water	Rainbow trout (Fw)	6 months	No data	PCB - 0.12 (liver) Dieldrin - not detectable (liver) Hexachlorobezene - 0.14 (liver) Pentachlorobenzene - 0.04 (liver)	Very small but significant increase in blood glucose and decreases in hematocrit and hemoglobin
Thomas et al. 1980	No 2. fuel oil	Fish (M)	3 hr	200-300 TN‡	100 TN‡ (muscle) 150 TN‡ (brain) 300 TN‡ (liver)	Increases in glucose, cholesterol, and cortisol
			12-288 hr	100 TN‡	10 TN‡ (muscle) 600 TN‡ (brain) 100 TN‡ (liver)	Return of serum parameters to control levels after 12 hr
Levitan and Taylor 1979	Naphthalene	Killifish (M)	12 hr	4000	12-23	Increase in serum cortisol
Dillon 1983	Dimethylnaphthalene	Grass shrimp (M)	32 days	0.24 µg/g wet food	4.12-4.32	No effect on blood copper concentrations.

‡ TN = total napthalenes.

Table 10

Frequency of Type of Biological Response Parameter Examined in the Reviewed Papers

Biological Response Parameters	Number of Entries	Percent of Total
Organismic parameters:		
Growth	46	22
Reproduction	33	15
Morphology/histology	32	15
Behavior	24	11
Metabolism	20	9
Osmotic/ionic regulation	7	3
Subtotal	160	75
Biochemical parameters:		
Enzymes	22	10
Biochemical composition	20	9
Blood chemistry	12	6
Subtotal	54	25
Total	214	100

Table 11

Highest No Effects Concentration (HNEC) and Lowest Effects Concentration (LEC) Grouped by Class of Contaminent

Contaminant	Value*	Concentration - µg/g, wet weight HNEC	LEC
All chlorinated hydrocarbons	$\bar{x}$	16.4	14.4
	±SD	±27.0	±36.8
	CV	162%	256%
	Range	0.08-112	0.02-180
	n	45	62
Polychlorinated biphenyls only	$\bar{x}$	35.8	45.3
	±SD	±37.1	±65.4
	CV	104%	144%
	Range	0.08-110	0.31-180
	n	16	15
All heavy metals	$\bar{x}$	31.0	43.5
	±SD	±62.0	±100
	CV	200%	230%
	Range	0.05-300	0.005-450
	n	52	49
Cadmium only	$\bar{x}$	38.2	64.8
	±SD	±66.0	±139
	CV	173%	214%
	Range	0.05-225	0.005-450
	n	22	22
Mercury only	$\bar{x}$	6.82	21.6
	±SD	±5.80	±36.0
	CV	85%	167%
	Range	2.7-16	1.1-150
	n	11	16
Petroleum Hydrocarbons	$\bar{x}$	117	127
	±SD	±181	±261
	CV	155%	206%
	Range	5.26-700	0.47-1000
	n	16	26

* Notations used in this column are defined as follows: $\bar{x}$ = mean; SD = standard deviation; CV = coefficient of variation (100 SD/$\bar{x}$); n = number of papers.

Table 12

Relative Sensitivity of Biological Responses to all Contaminants as Measured by the Lowest Effects Concentration (LEC)

Biological Response Parameters	LEC, µg/g, Wet Weight*				
	x	±SD	CV, %	Range	n
Organismic parameters:					
Growth	66.9	±187	280	0.07-450	35
Reproduction	40.4	±106	262	0.04-450	20
Morphology/histology	7.0	±12.3	176	0.11-50	21
Behavior	69.3	±164	237	0.02-280	20
Metabolism	76.5	±208	272	0.25-700	11
Osmotic/ionic regulation	26.0	±21.7	83.5	1.0-40.0	3
Total	50.9	±149	290	0.02-700	110
Biochemical parameters:					
Enzymes	26.1	±31.7	121	3.0-83.8	6
Biochemical composition	44.7	±85.0	190	0.22-280	11
Blood chemistry	18.9	±27.2	144	0.11-83.8	10
Total	31.3	±57.9	185	0.11-280	27

* Notations used as column headings are as follows: $\bar{x}$ = mean; SD = standard deviation; CV = coefficient of variation (100 SD/$\bar{x}$); n = number of papers.